COVID-19 PNEUMONIA ASSOCIATED STEMI: A CASE REPORT

Danielle L Zambrano DO[1]
Alexandra M Cruz DO[1]
1. Department of Internal Medicine, Philadelphia College of Osteopathic Medicine, Philadelphia, PA

INTRODUCTION

▪Coronavirus 19 (COVID 19) is a viral infection which has various forms of manifestations, ranging from asymptomatic to severe acute respiratory distress syndrome. Commonly presenting as a viral pneumonia, many patients have been hospitalized for acute hypoxic respiratory failure as a result. In certain patients, the cardiovascular system may be involved to some extent. Leakage of cardiac enzymes may be detected as a result of demand ischemia. We present an interesting case of COVID-19 associated ST segment elevation myocardial infarction.

CASE REPORT

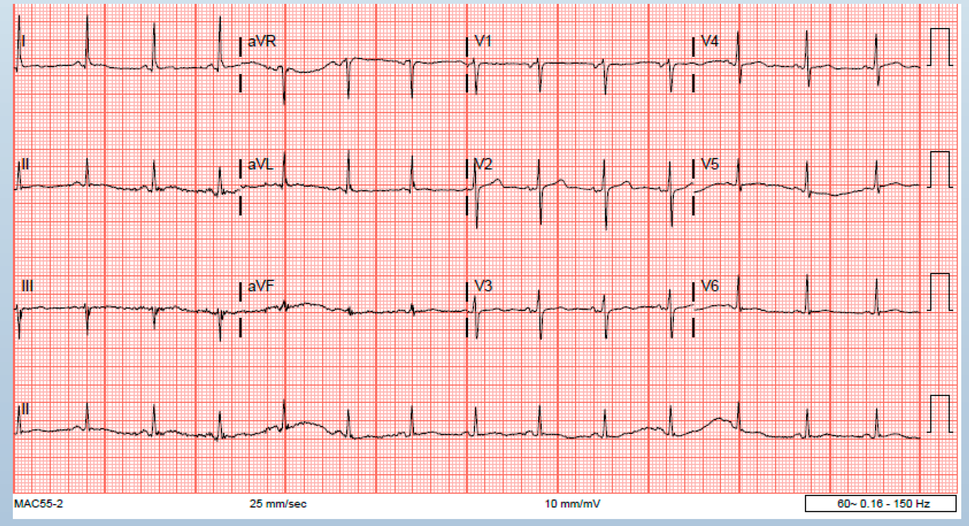
59 year old unvaccinated male with PMHx HTN, HLD, T2DM who presented from home for progressively worsening shortness of breath. Patient developed symptoms one week after a COVID exposure after which he tested positive for COVID. Initially, patient was discharged home due to hemodynamic stability; however he returned to the hospital one week later when his shortness of breath continued to worsen. On admission, patient was hypoxic to 85% on room air, requiring 5 L nasal cannula on admission. He was admitted to the general medical floors for acute hypoxemic respiratory failure secondary to COVID pneumonia.

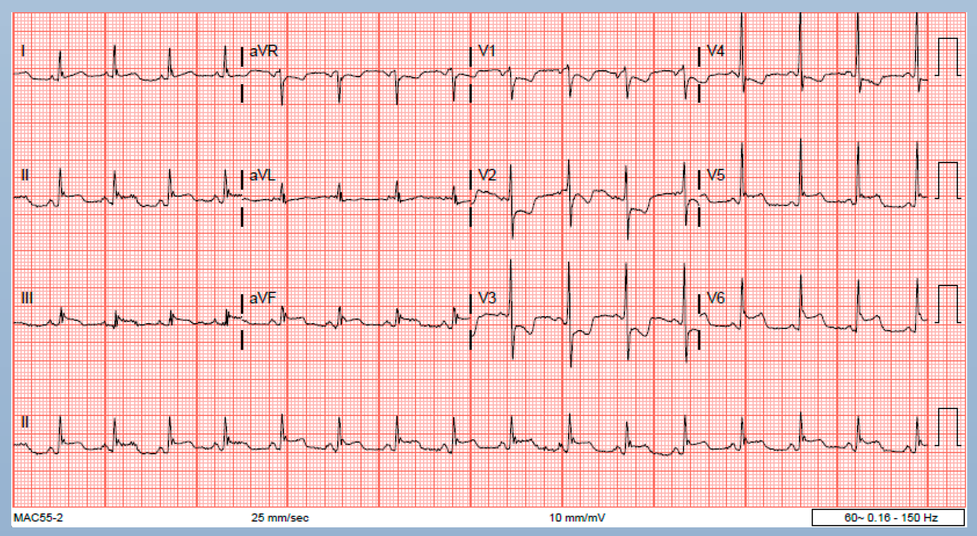
Patient was given one dose of Tocilizumab, a five day course of Remdesivir and steroids. Doxycycline was added due to concerns for superimposed bacterial infection. Oxygen requirements continued to increase throughout patient's hospitalization. Patient could not tolerate high flow nasal cannula due to discomfort. On the 6th day of patient's hospitalization, rapid response was called due to increased work of breathing and worsening hypoxemia for which he was placed on nonrebreather mask which was then escalated to BiPAP which temporarily provided symptomatic relief. EKG obtained during rapid response did not reveal any acute ischemic changes. Patient endorsed pleuritic chest pain which was reproducible to palpation.

Approximately 1.5 hours after initial rapid response was called, patient developed acute onset chest pain. Repeat EKG obtained revealed ST segment elevations in lateral leads V5 and V6 with reciprocal changes in leads V1 and V4. Troponin from initial rapid response was 0.16 ng/mL, repeat troponin had increased to 1.10 ng/mL. Bedside echocardiogram revealed low normal LV systolic function, with a hypokinetic lateral wall. Cardiology was consulted and recommended emergent transfer for cardiac catheterization. Patient was transferred to a tertiary care facility where he underwent cardiac catheterization. Patient was found to have thrombotic occlusion of the mid left circumflex artery. Drug-eluting stent was successfully placed. Post procedure, patient was started on aspirin, plavix, atorvastatin, and heparin drip. He was later transitioned to therapeutic lovenox.

DISCUSSION

▪The following criteria for ST-elevation myocardial infarction: new ST-segment elevation at the J point in 2 contiguous leads with the cutoff point as greater than 0.1mV in all leads other than V2 or V3, In leads V2-V3. Patients with a pre-existing left bundle branch block can be further evaluated using Sgarbossa's criteria: ST-segment elevation of 1mm or more that is concordant with the QRS complex, ST-segment depression of 1mm or more in lead V1,V2, or V3, StT-segment elevation of 5mm or more that is discordant with the QRS complex. Prompt restoration of myocardial blood flow is essential to optimize myocardial salvage and to reduce mortality.





CONCLUSION

- This patient presented with acute hypoxemic respiratory failure due to COVID-19 infection subsequently developed a prothrombotic state resulting in STEMI. This patient had a STEMI secondary to thrombotic event in angiographically normal coronary arteries, most likely a prothrombotic state secondary to COVID-19 infection. Regardless of COVID status, managmentment of ST segment myocardial infarction remains the same in COVID positive patient as in a COVID negative patient. The mainstay of treatment remains cardiac reperfusion. In conclusion, patient's with COVID-19 are at higher risk of life threatening prothrombotic events.

REFERENCES

Anderson JL, Karagounis LA, Califf RM. Metaanalysis of five reported studies on the relation of early coronary patency grades with mortality and outcomes after acute myocardial infarction. Am J Cardiol 1996; 78:1.
Sgarbossa EB, Pinski SL, Barbagelata A, Underwood DA, Gates KB, Topol EJ, Califf RM, Wagner GS. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med. 1996 Feb 22;334(8):481-7.
Smith SW, Dodd KW, Henry TD, Dvorak DM, Pearce LA. Diagnosis of ST-elevation myocardial infarction in the presence of left bundle branch block with the ST-elevation to S-wave ratio in a modified Sgarbossa rule. Ann Emerg Med. 2012 Dec;60(6):766-76.
Stub D, Smith K, Bernard S, Nehme Z, Stephenson M, Bray JE, Cameron P, Barger B, Ellims AH, Taylor AJ, Meredith IT, Kaye DM., AVOID Investigators. Air Versus Oxygen in ST-Segment-Elevation Myocardial Infarction. Circulation. 2015 Jun 16;131(24):2143-50.